physicians' responsibility and obligations are still being negotiated.

Physicians are correct in being apprehensive that what would seem a welcome relief from malpractice insurance costs may instead be trouble. If legislative or regulatory pressure produces hospital liability for medical staff decisions, only a fundamental change in the relationship between hospital and physician staff will permit the continuing influence of physician judgment to be a dominant factor in the appraisal and assurance of quality of care and in the evaluation, counseling, and credentialing of the medical staff.

Concerned physicians might well begin to explore the organizational possibilities that differ from the conventional governing board–administrator–medical staff arrangement. Loosely affiliated staff status may no longer be tenable when changes in law interpose a hospital accountability between physician and patient. Certainly the resources of medical societies should be used to call on legal and organizational experts to consider changes in the hospital "culture" that will best maintain the role of physicians in quality-of-care decisions and in the oversight of quality of care if hospital liability comes to pass.

What organizational changes will best meld physicians' interests with those of the hospital, so that physicians' professional autonomy can continue, and physicians, not administrators, more effectively monitor the work of medical staff? Can a new medical staff relationship promote physician loyalty and concern for hospital well-being while ensuring physician participation in the collective actions and voice of the hospital?

The introduction of "hospital culture" into the discussion is neither irreverent nor irrelevant. Proponents of corporate medicine note the "social insularity of the physician group," that "the physician profession-based culture may 'clash' with corporate culture," that "the conduct of physicians must be modified," that hospitals, among others, "are presently attempting to alter physician practice patterns," and that "managers can then establish organizational environments that effectively reduce the tension between cultures of the physician and the organization."[2]

I urge physicians to be adequately prepared to have an equal share in developing and guiding these new organizational environments. Now, while hospital liability is being discussed, and before it is implemented, is the time for physicians to consult with organizational and legal specialists who may best design a system that fosters the best provision of care, staff autonomy, oversight of colleagues' work, and accountability to patients and the community.

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Cocaine Toxicity in Glycogen Storage Disease

TO THE EDITOR: Cocaine has been implicated as a skeletal muscle toxin, producing nontraumatic rhabdomyolysis[1-3] or exacerbation of muscle weakness.[4] We report a case of a patient with previously diagnosed but asymptomatic glycogenosis who had prolonged respiratory muscle insufficiency after inhaling crack cocaine. This is the first report of such a case.

Report of a Case

The patient, a 26-year-old woman, was diagnosed at age 3 with glycogenosis type IIIA by muscle and liver biopsy in which the specimen showed an absence of glycogen debranching enzyme in both tissues. Before the current admission to hospital, she had had no clinical signs of muscle weakness, but had mildly elevated creatine kinase levels. She was admitted to the University Medical Center, Las Vegas, Nevada, in respiratory distress after several days of smoking crack cocaine. She was 16 weeks' pregnant. Her clinical condition worsened, necessitating intubation and mechanical ventilation. At this time she had shoulder girdle weakness, being unable to abduct her arms against minimal pressure. The chest radiograph showed bilateral, diffuse alveolar infiltration. Echocardiography revealed good contractile function. Her pulmonary capillary wedge pressure was normal. The patient's serum chemistry values were notable for a potassium level of 3.3 mmol per liter (normal, 3.5 to 5.5), aspartate aminotransferase 116 units per liter (normal, 5 to 35), creatine kinase 2,000 units per liter (normal, 35 to 230) without elevated levels of the myocardial-specific isoenzyme, triglycerides 3.10 mmol per liter (275 mg per dl [normal, 0.34 to 1.70]), and cholesterol 6.10 mmol per liter (236 mg per dl [normal, 3.65 to 5.20]). Serum levels of calcium, phosphorus, and magnesium were within normal limits. The urine myoglobin test was negative.

The patient's cocaine-induced pulmonary edema abated notably within two days of intubation, but she could not be weaned from mechanical ventilation. Attempts at weaning showed that she was unable to generate a static inspiratory pressure of 20 cm of water and maintained a respiratory rate of 30 to 40 breaths per minute to avoid hypercarbia. She remained on mechanical ventilation for 11 days before finally being able to breathe effectively on her own. Her creatine kinase serum level decreased to 1,150 units per liter before weaning from mechanical ventilation was possible.

The patient had a normal neonate by cesarean section after a pregnancy of nine months and was later lost to follow-up.

Discussion

Cocaine use unmasked the subclinical skeletal muscle weakness in our patient. Glycogenosis type III (Cori's disease) is an autosomal recessive glycogen storage disease caused by an inherited deficiency of glycogen debranching enzyme, with a resultant accumulation of

glycogen in the liver alone, type IIIB, or in liver and muscle tissue, type IIIA.[5-7] Persons with this condition can present in infancy with hypoglycemia, hepatomegaly, or, rarely, cardiac failure. Those with adult-onset disease, however, can be completely asymptomatic or have a progressive weakness of skeletal musculature.[6] Serum levels of muscle enzymes such as creatine kinase can be elevated even in asymptomatic persons with adult-onset glycogenosis type III. Others, such as our patient, with a deficiency of debranching enzyme in muscle are more likely to have myopathy and cardiomyopathy.[7]

Cocaine use has been responsible for nontraumatic rhabdomyolysis in several reports in which urine myoglobin was present,[1-3] which suggests that cocaine may have a direct lytic effect on skeletal muscle or that it produces muscle ischemia by vasospasm. It may also reduce myocyte excitability and subsequent tension generation[2] and has been implicated as a cause of impairment of neuromuscular transmission in a patient with myasthenia gravis.[4] Thus, several mechanisms by which cocaine exerts deleterious effects on skeletal muscle are likely. Cocaine abuse reduced the threshold whereby our patient's muscle weakness became clinically apparent, manifesting as respiratory muscular insufficiency.

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Advances in Cutaneous Laser Surgery

TO THE EDITOR: We were astonished to read the epitome in the April 1993 issue entitled "Laser Treatment of Skin Lesions" by Thomas R. Stevenson, MD.[1] The epitome was intended to be an update on firmly established treatment methods that are of clinical importance in laser surgery of cutaneous lesions. Unfortunately, it ignored some of the most important advances that have occurred in laser surgery in the past five years, specifically the use of the pulsed-dye laser for treating vascular lesions and Q-switched lasers for removing tattoos. Both of these lasers are based on the principles of selective photothermolysis as developed by Anderson and Parrish.[2]

The basic principles of this technique are to use a wavelength that will be selectively absorbed by the chromophore target of the lesion to be treated and to use a pulse width that is short enough to prevent nonselective thermal damage to the surrounding tissue. The flashlamp-pumped pulsed-dye laser, with a wavelength of 585 nm and a pulse width of 450 μsec, selectively targets vascular lesions, enabling the treatment of these lesions without the risk of scarring. Stevenson states that young children with port-wine stains are not good candidates for treatment because of the risk of scarring. This is indeed true if the argon laser is being used. The use of the pulsed-dye laser has revolutionized this therapy, however, as this laser is specifically designed for treating vessels that occur in infants and children having these capillary malformations. With this laser, the younger the child, the more effective is the treatment.[3] Infants can be readily treated without the risk of scarring. Early treatment of capillary hemangiomas is also effective with this laser, which will arrest the proliferative stage of the lesion, induce an involutional stage, and may prevent complications, scarring, and emotional distress associated with these lesions.[4]

Although both the neodymium:yttrium-aluminum-garnet (Nd:YAG) laser and the argon laser have been used successfully to treat capillary hemangiomas, the use of these lasers carries with it a substantial risk of scarring as a consequence of the nonspecific thermal damage that occurs.

Although the carbon dioxide laser has been used for many years to remove tattoos, this treatment invariably results in scarring, as the target of the laser is the skin containing the tattoo rather than the tattoo itself. No specific reaction occurs with the tattoo pigment, but rather a nonspecific thermal effect results in a slough of the tissue containing the pigment. In contrast, the Q-switched lasers—the Q-switched ruby,[5] Q-switched alexandrite,[6] and Q-switched Nd:YAG lasers—are targeted specifically to the tattoo pigment and result in fragmentation of the pigment into small particles that are picked up by macrophages and removed from the tissue.[7] No direct tissue interaction occurs, and as a consequence, tattoo removal can be accomplished without producing scar tissue. These lasers represent a tremendous technologic advance.

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